UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
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August 11, 2008

Reply To: OEA095

MEMORANDUM

SUBJECT: **Proposed Risk Assessment Approach for Evaluating Potential Risks From Consuming Breast Milk**
Portland Harbor Superfund site

FROM: Government Risk Assessment Team

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TO: Lower Willamette Group

INTRODUCTION

One of the remaining questions we have for the Portland Harbor Superfund human health risk assessment (HHRA) is how to evaluate potential risks to infants from consuming chemicals in breast milk as a result of maternal exposure from eating contaminated fish and other biota. EPA and its partners evaluated the feasibility of conducting a risk assessment based on exposure to breast milk using EPA's *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities*[1] (Combustion Guidance), *Exposure Factors Handbook*[2], *Child-Specific Exposure Factors Handbook*[3], and examples from other hazardous waste sites, such as the Housatonic river project in Massachusetts[4]. We determined that it is feasible to include exposure to breast milk in the Portland Harbor HHRA, and that this is an important exposure pathway.

To assist the Lower Willamette Group in incorporating the breast milk consumption pathway into the HHRA, we prepared this memorandum to present the relevant exposure and risk equations, and exposure and toxicity parameters (summarized in Table 1). We include example calculations using total polychlorinated biphenyl (PCB) Aroclors to show how the various equations in EPA's combustion guidance can be modified to focus on the fish consumption pathway. The actual risk assessment should include all relevant chemicals, such as total PCBs (from Aroclors or congeners), 2,3,7,8-TCDD equivalents (from chlorinated dibenzo-p-dioxins, chlorinated dibenzofurans, and dioxin-like PCB congeners, evaluating each chemical class separately and collectively as the sum of all dioxin-like chemicals), and DDT and its degradation products.

Generally, risk assessments are limited to an evaluation of risk, and do not consider comparative risks or benefits. For example, eating fish is health beneficial compared with eating other protein sources. Public health agencies commonly address the health tradeoffs of eating contaminated fish, but the issue is not typically discussed in a Superfund risk assessment. For breast feeding, however, the benefits to infants are so substantial that we consider it appropriate to discuss the issue in the risk assessment. We therefore will provide the Lower Willamette Group with a letter from the Oregon Environmental Health Assessment Program (EHAP) that presents the risks and benefits of consuming contaminated breast milk. We request that this information be included in the Portland Harbor risk assessment.

PROPOSED RISK ASSESSMENT APPROACH

Exposure Assessment

We mainly relied on the equations presented in the EPA combustion guidance document[1], modified to make the equations no longer specific to chlorinated dioxins or the inhalation pathway, and instead make them appropriate for fish consumption. The key concept is that the concentration of a chemical in milk can be calculated from the long-term body burden in the mother. This is consistent with the information presented in the Agency for Toxic Substances Disease Registry (ATSDR) *Toxicological Profile for Polychlorinated Biphenyls*[5].

We start with the average daily intake of chemicals from fish consumption (modified from Table C-1-4 of the Combustion Guidance[1]):

$$ADD_{mother} = \frac{C_{fish} \times IR_{fish} \times CF \times F_{fish}}{BW_{af}}$$

Where:

ADD_{mother}	=	Average daily dose to mother (mg/kg/day)
C_{fish}	=	Chemical concentration in fish (assume 1 mg/kg for PCBs)
IR_{fish}	=	Ingestion rate of fish (subsistence rate of 142.4 g/day)
CF	=	Conversion factor (0.001 kg/g)
F_{fish}	=	Fraction of fish contaminated (1)
BW_{af}	=	Body weight (66 kg for average adult female)

The ingestion rate used in the example is that being used in the Portland Harbor HHRA for fishers subsisting on resident fish. The site risk assessment should include all of the other relevant fish consumption rates being used for the Portland Harbor HHRA. The fish consumption rate is an annualized rate (*i.e.*, it includes the assumption that fish are eaten throughout the year, so exposure frequency, exposure duration, and averaging time are not included in the equation). Loss of chemicals during cooking, which has been considered at other sites, is not included in EPA's Combustion Guidance. However, cooking loss will be addressed in the uncertainty section of the Portland Harbor HHRA

Risk Characterization. For body weight, we consider it appropriate to use the average female weight of 66 kg, rather than the guidance value of 70 kg (average adult weight).

For this example, the calculations are performed assuming a total PCB concentration of 1 mg/kg in whole-body tissue. This value is within the range of PCB concentrations measured in Portland Harbor resident fish composites and was chosen primarily for illustrative purposes. The actual risk assessment should use chemical concentrations appropriate for the various species of fish sampled.

$$ADD_{mother} = 1\text{ mg/kg} \times 142.4\text{ g/day} \times 0.001\text{ kg/g} \times 1 / 66\text{ kg} = 0.0022\text{ mg/kg/day}$$

EPA has found that dietary intake of PCBs during pregnancy and lactation is only weakly correlated with PCB concentrations in human milk. The more important determinant is long-term consumption of PCBs. The following equation is used to calculate the PCB concentration in milk fat.

$$C_{milkfat} = \frac{ADD_{mother} \times h \times f_1}{\ln(2) \times f_2}$$

Where:

$C_{milkfat}$ = PCB concentration in milkfat (mg/kg-lipid)
ADD_{mother} = Average daily dose to mother (mg/kg/day)
h = Half-life of PCB (7 years = 2555 days)
f_1 = Fraction of ingested PCB stored in fat (0.9)
f_2 = Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)

$$C_{milkfat} = \frac{0.0022\text{ mg/kg-totalBW/day} \times 2555\text{ days} \times 0.9}{0.693 \times 0.3\text{ (kg-lipidBW/kg-totalBW)}}$$

$$= 24\text{ mg/kg-lipid}$$

The equation was modified from Table C-3-1 of the Combustion Guidance[1], and is consistent with equations 1 through 3(b) in Section 3.4.4.2 of the ATSDR *Toxicological Profile*[5]. The equation is for steady-state conditions, and therefore we assume that maternal intake occurs over a time-period greater than the PCB half-life. We also assume that PCB concentrations in breast milk reflect the maternal body burden. For a derivation of the equation for $C_{milkfat}$, see Attachment A.

Average daily doses to the infant are calculated separately for carcinogenic and noncarcinogenic effects. For carcinogenic effects, the average daily dose is the following (modified from Table C-3-2 of the Combustion Guidance[1]):

$$ADD_{ca\text{-}infant} = \frac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{AT_i \times BW_i}$$

Where:

$ADD_{ca\text{-}infant}$ = Average daily dose for breast-feeding infant (mg/kg/day)

$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)
IR_{milk} = Ingestion rate of breast milk (0.69 kg/day)
f_3 = Fraction of breast milk that is fat (0.04)
f_4 = Fraction of ingested PCB that is absorbed (0.9)
ED_i = Exposure duration of breast-feeding infant (1 year)
EF_i = Exposure frequency of breast-feeding infant (365 days/year)
AT_i = Averaging time – carcinogen (70 years x 365 days/year)
BW_i = Body weight of breast-feeding infant (9.4 kg)

$$ADD_{ca\text{-}infant} = \frac{24 \text{ mg/kg-lipid} \times 0.69 \text{ kg/day} \times 0.04 \times 0.9 \times 1 \text{ yr} \times 365 \text{ day/yr}}{70 \text{ yr} \times 365 \text{ day/yr} \times 9.4 \text{ kg}}$$

$$= 0.00091 \text{ mg/kg/day}$$

For non-cancer effects, the average daily dose is the following (modified from Table C-3-2 of the Combustion Guidance[1]):

$$ADD_{nc\text{-}infant} = \frac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{AT_{nc} \times BW_i}$$

Where:
$ADD_{nc\text{-}infant}$ = Average daily dose for breast-feeding infant (mg/kg/day)
$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)
IR_{milk} = Ingestion rate of breast milk (0.69 kg-milk/day)
f_3 = Fraction of breast milk that is fat (0.04 kg-lipid/kg-milk)
f_4 = Fraction of ingested PCB that is absorbed (0.9)
ED_i = Exposure duration of breast-feeding infant (1 year)
EF_i = Exposure frequency of breast-feeding infant (365 days/year)
AT_{nc} = Averaging time – non-carcinogen (= ED_i x EF_i)
BW_i = Body weight of breast-feeding infant (9.4 kg)

$$ADD_{nc\text{-}infant} = \frac{24 \text{ mg/kg-lipid} \times 0.69 \text{ kg-milk/day} \times 0.04 \text{ kg-lipid/kg-milk} \times 0.9 \times 1 \text{ yr} \times 365 \text{ day/yr}}{1 \text{ yr} \times 365 \text{ day/yr} \times 9.4 \text{ kg}}$$

$$= 0.063 \text{ mg/kg/day}$$

Toxicity Assessment

A hierarchy for selecting human health toxicity factors for use in Superfund risk assessments is provided in EPA's *Human Health Toxicity Values in Superfund Risk Assessments*[6]. The recommended toxicity hierarchy is as follows:

Tier 1 – EPA's Integrated Risk Information System (IRIS)

Tier 2 – EPA's Provisional Peer Reviewed Toxicity Values (PPTRVs)

Tier 3 – Other Toxicity Values

For estimating carcinogenic risk for an infant from breast feeding, the PCB cancer slope factor presented in IRIS was used 2 $(mg/kg/day)^{-1}$. This value is applied to total PCBs in the example calculation.

For estimating PCB non-cancer hazards for an infant from breast feeding, a toxicity value from a Tier 3 toxicity source, the Agency for Toxic Substances and Disease Registry (ATSDR) was used rather than the IRIS Reference Dose (RfD). The RfD for PCBs in IRIS, 2×10^{-5} mg/kg/day, was established for chronic exposures of 7 years to a lifetime. For breast milk exposure, it is assumed that exposure to an infant occurs for one year. IRIS defines a subchronic exposure as exposure up to 10% of an average human lifespan. As there is no RfD for subchronic exposure for PCBs in IRIS, following EPA's toxicity hierarchy, the ATSDR's PCB minimal risk level (MRL)of 3×10^{-5} mg/kg/day for intermediate-duration (subchronic) oral exposure was chosen. ATSDR defines intermediate-duration exposure as two weeks to one year. The intermediate-duration MRL was derived using data on monkeys that approximated exposure during breastfeeding. For this reason, it is a more appropriate toxicity value than the IRIS chronic PCB RfD (which is equal to the chronic MRL).

Risk Characterization

Calculated Cancer Risk to Infants

Using the standard risk characterization equations, excess lifetime cancer risk and non-cancer hazards are calculated separately. Excess lifetime cancer risk is approximated by:

$$ELCR_{infant} = ADD_{ca\text{-}infant} \times SF_o$$

Where:

$ELCR_{infant}$	=	Excess lifetime cancer risk to infant from breast feeding
$ADD_{ca\text{-}nfant}$	=	Average daily dose for breast-feeding infant (mg/kg/day)
SF_o	=	Cancer slope factor – oral [2 $(mg/kg/day)^{-1}$ for total PCBs]

$$ELCR_{infant} = 0.00091 \text{ mg/kg/day} \times 2\ (\text{mg/kg/day})^{-1} = 2 \times 10^{-3}$$

Calculated Non-Cancer Risk to Infants

The non-cancer hazard quotient is:

$$HQ_{infant} = \frac{ADD_{infant}}{MRL}$$

Where:

HQ_{infant}	=	Hazard quotient for breast-feeding infant
MRL	=	Non-cancer subchronic MRL (3×10^{-5} mg/kg/day for total PCBs)

Using the intermediate-duration MRL, the calculated hazard quotient is:

$$HQ_{infant} = 0.063 \text{ mg/kg/day} / 3 \times 10^{-5} \text{ mg/kg/day} = 2{,}100$$

<u>Comparison of Calculated Risks with Acceptable Levels</u>

Using the approach presented in this memorandum, the excess lifetime cancer risk is approximately 2×10^{-3} for an infant consuming total PCBs in breast milk for one year. This is above the cancer risk range of 10^{-4} to 10^{-6}, the target range within which the EPA strives to manage risks as part of a Superfund Cleanup. The acceptable excess lifetime cancer risk under Oregon Department of Environmental Quality rules[7] is 1×10^{-6}.

For non-cancer effects, the calculated hazard quotient is 2,100. For hazard quotients above 1, unacceptable exposures may be occurring and there may be concern for potential non-cancer effects. Under Oregon rules, the acceptable hazard quotient is 1. Generally, the greater the magnitude of the hazard quotient above 1, the greater the level of concern for non-cancer health effects.

The calculated cancer risks and non-cancer hazards are based on a total PCB concentration in whole-body resident fish composites of 1 mg/kg. Although this concentration was used as a convenient value to demonstrate the calculations, it is within the range of total PCBs in resident fish composites in the initial study area of the lower Willamette River. Reasonable maximum exposure whole-body smallmouth bass concentrations of PCBs by river mile ranged from 0.25 mg/kg to 4.5 mg/kg. The site-wide reasonable maximum exposure PCB concentration in whole-body common carp is 5.9 mg/kg, which would result in a hazard quotient of 12,000 given the same exposure assumptions used for smallmouth bass. Because the calculated excess lifetime cancer risk and hazard quotient are considerably above acceptable levels, we conclude that infant exposure to chemicals in breast milk is an important pathway for the Portland Harbor HHRA.

The risk levels associated with the breast feeding pathway are similar to, or well above those associated with direct consumption of fish by adult subsistence fishers. For consumption of whole-body smallmouth bass, the calculated risks presented in Appendix F, Table 5-35 of the Portland Harbor Round 2 Report[8] range from excess lifetime cancer risks of 4×10^{-4} to 8×10^{-3}, and the hazard quotients range from 30 to 500 for adult fish consumption at 142 grams per day.

UNCERTAINTY EVALUATION

Following standard guidance, the risk assessment for this pathway should include an evaluation of the associated uncertainties. During our evaluation of this pathway, we considered the following.

<u>Exposure Assessment</u>

The only exposure to infants evaluated was consumption of breast milk. We did not consider other potential exposure routes, such as transplacental transfer of PCBs from mother to fetus during pregnancy.

ATSDR considers exposure of one year or more to be chronic exposure. However, EPA's Superfund program defines seven years or more as chronic exposure[9]. To evaluate chronic exposure, we included an alternative child exposure with one year of breast-feeding exposure, and six years of fish consumption, for a total child exposure period of seven years.

$$ADD_{child} = \frac{\dfrac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{BW_i} + \dfrac{C_{fish} \times IR_{fish} \times CF \times ED_c \times EF_c}{BW_c}}{AT}$$

Where:

ADD_{child}	=	Average daily dose for breast-feeding and fish-eating child (mg/kg/day)
$C_{milkfat}$	=	Concentration of chemical in milk fat (mg/kg-lipid)
C_{fish}	=	Chemical concentration in fish (assume 1 mg/kg for PCBs)
IR_{milk}	=	Ingestion rate of breast milk (0.69 kg-milk/day)
IR_{fish}	=	Ingestion rate of fish (60 g/day)
f_3	=	Fraction of breast milk that is fat (0.04 kg-lipid/kg-milk)
f_4	=	Fraction of ingested PCB that is absorbed (0.9)
ED_i	=	Exposure duration of breast-feeding infant (1 year)
EF_i	=	Exposure frequency of breast-feeding infant (365 days/year)
ED_c	=	Exposure duration of child (6 years)
EF_c	=	Exposure frequency child (365 days/year)
BW_i	=	Body weight of breast-feeding infant (9.4 kg)
BW_c	=	Body weight of child (15 kg)
AT	=	Averaging time (non-carcinogen = 7 years infant and child) (carcinogen = 70 years)

Using the approach to evaluate chronic exposure (1 year as an infant, 6 years as a child), the calculated child exposure to PCBs for carcinogenic risk is

$$ADD_{ca\text{-}child} = 0.0012 \text{ mg/kg/day}$$

This ADD for cancer effects for the 7 year infant and child exposure is about 1.3 times the dose to the infant alone.

The calculated child exposure to PCBs for non-carcinogenic risk is

$$ADD_{nc\text{-}child} = 0.012 \text{ mg/kg/day}$$

This ADD for non-cancer effects for the 7 year infant and child exposure is about 0.2 times the dose to the infant alone.

Toxicity Assessment

The PCB RfD is based on LOAELs developed from studies on monkeys. The health effects included inflammation of glands in the eye, distorted growth of finger and toe nails, and decreased antibody responses. The uncertainty factors used in the derivation of the human health RfD total 300, applied to an animal LOAEL of 0.005 mg/kg/day. The calculated HQ from consumption of breast milk is from 2 to 10 times greater than the uncertainty factor.

As previously discussed, the ATSDR intermediate-duration PCB MRL was used to estimate non-cancer impacts for the infant exposed for one year rather that the PCB RfD in IRIS. In its Combustion Guidance[1], EPA considered it appropriate to apply the chronic RfD to one year of exposure to breast milk, at least for screening purposes. Application of the chronic RfD to one year of exposure may also be appropriate considering the potential sensitivity of infants to adverse health effects.

Risk Characterization

Using the longer exposure period of seven years for a child, the calculated ELCR is essentially the same value as that calculated for one year of infant exposure.

$$ELCR_{child} = 0.0012 \text{ mg/kg/day} \times 2 \text{ (mg/kg/day)}^{-1} = 2 \times 10^{-3}$$

Where:

$ELCR_{child}$ = Excess lifetime cancer risk to infant from breast feeding and child from eating fish

Using the chronic RfD for PCBs instead of the intermediate-duration MRL for one year of breast feeding, the calculated hazard quotient is:

$$HQ_{infant} = 0.063 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 3{,}200$$

Using the chronic RfD along with the longer exposure period of seven years for a child, the calculated hazard quotient is:

$$HQ_{child} = 0.012 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 600$$

These hazard calculations modified to take into account chronic exposure are 1.5 to 0.3 times the subchronic HQ of 2,100.

Body Burden Reductions

We also looked at the reduction in body burden of PCB during a year of breast feeding to see if that could result in reduced concentrations in breast milk. If the concentration in milk fat (C_{milk} = 24 mg/kg-lipid) is equivalent to the concentration in other tissues (C_{lipid}), then the body burden in the mother is:

$$C_{lipid} \times BW_{af} \times f_2 =$$

$$24 \text{ mg/kg-lipid} \times 66 \text{ kg-BW} \times 0.3 \text{ kg-lipid/kg-BW} = 480 \text{ mg PCB}$$

The loss of mass during one year of breast feeding is:

$$IR_{milk} \times C_{milkfat} \times f_3 \times 365 \text{ days} =$$

$$0.69 \text{ kg/day} \times 24 \text{ mg/kg-lipid} \times 0.04 \text{ kg-lipid/kg-milk} \times 365 \text{ days} = 240 \text{ mg PCB}$$

This implies that a mother will lose approximately half of her PCB body burden (240 mg / 480 mg) during a year of breast feeding, assuming that there is no additional consumption of contaminated fish during this period. This simplistic evaluation is consistent with EPA's determination (summarized in the GE/Housatonic report[4]) that there will be a 20 percent reduction of PCBs in the mother every three months. Over a year, this would correspond to a reduction of $1 - (1 - 0.2)^4 = 0.6$, or a 60 percent reduction in PCB mass after one year. The reduction in mass (and concentration) averaged over the course of the year would be about half of this value. If we assume that the PCB concentration in breast milk reduces to one-half the original value in one year, then the average concentration consumed by the infant over the year would be about three-quarters of the original concentration. The corresponding risk and hazard calculations would be lower by this amount. Refining the calculations to include this reduction in mass would reduce the calculated hazard quotient by a factor of about 1.3.

Relative Exposure

At other sites, including the Housatonic River site[4], EPA presented the potential risks from breast milk consumption as a ratio to background risk rather than as an excess lifetime cancer risk or hazard quotient. The background total PCB concentration used for the Housatonic River site is 0.32 mg/kg-lipid in milk. Using the assumed total PCB concentration of 1 mg/kg in Portland Harbor fish tissue and the assumed subsistence fish consumption rate, the calculated total PCB concentration in breast milk is 24 mg/kg-lipid. As an alternative presentation of risk in the uncertainty section, this result can be discussed as corresponding to a risk 75 times that of the background concentrations used for the Housatonic River site.

Fish Advisory

EPA is aware that in the lower Willamette River, consumption of resident fish by lactating mothers is already discouraged by the PCB fish advisory[9]. The Oregon Department of Human Services (DHS) advisory states that:

> Women of childbearing age, particularly pregnant or breastfeeding women, children and people with weak immune systems, thyroid or liver problems, should avoid eating resident fish from Portland Harbor, especially carp, bass and catfish.

For this reason, there may currently be limited infant exposure to breast milk contaminated as a result of consumption of resident fish in the lower Willamette River. In addition, DHS advice on preparing fish for consumption, including removing fat from fillets (rather than consuming whole-body fish), could substantially lower risks to fish consumers, and also subsequently to breast-feeding infants. However, the results presented here appear to quantitatively support the advisory, and indicate that there are potentially unacceptable risks by the breast-feeding pathway.

HEALTH CONSULTATION ON BREAST-FEEDING PATHWAY

EPA asked the Oregon Environmental Health Assessment Program (EHAP, formerly SHINE) to develop recommendations on how to address the potential health risks for infants exposed to PCBs in breast milk in the context of the many health benefits of breast feeding. EHAP's evaluation and recommendations are being reviewed by ATSDR, and will be provided in a separate letter to the Lower Willamette Group. A summary of the letter will be included in the final version of this memorandum.

ENDNOTES

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[1] U. S. EPA. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* (EPA 530-R-05-006, September 2005.
[2] U.S. EPA. *Exposure Factors Handbook.* National Center for Environmental Assessment, Office of Research and Development. August 1997.
[3] U.S. EPA. *Child-Specific Exposure Factors Handbook.* National Center for Environmental Assessment, Office of Research and Development. EPA-600-P-00-002B, Interim Report. September 2002.
[4] U.S. Army Corps of Engineers, U.S. EPA. *Human Health Risk Assessment, GE/Housatonic River Site, Rest of River,* Volume 1. February 2005.
[5] Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Polychlorinated Biphenyls* (Update, November 2000).
[6] U.S. EPA, Human Health Toxicity Values in Superfund Risk Assessments. OSWER Directive 9285.7-53, December 5, 2003.
[7] Oregon Administrative Rules, Chapter 340, Division 122, Section 0115.
[8] Lower Willamette Group. *Portland Harbor RI/FS Comprehensive Round 2 Site Characterization Summary and Data Gaps Analysis Report.* 21 February 2007.
[9] U.S. EPA. *Risk Assessment Guidance for Superfund*. Volume 1. Human Health Evaluation Manual (Part A). Interim Final. (EPA 540-1-89-002). December 1989.

Table 1
Parameters for Evaluation of Risk from Consuming Breast Milk

Parameter	Units	Description	Value[a]
ADD_{mother}	mg/kg/day	Average daily dose to mother	Calculated
$ADD_{ca\text{-}child}$	mg/kg/day	Average daily dose to child (cancer)	Calculated
$ADD_{nc\text{-}child}$	mg/kg/day	Average daily dose to child (non-cancer)	Calculated
C_{fish}	mg/kg	Chemical concentration in fish	Calculated from site data. Assume 1 for example.
IR_{fish}	g/day	Ingestion rate of fish	142.4 for subsistence fishers[b]
IR_{milk}	kg/day	Ingestion rate of breast milk	0.69
CF	kg/g	Conversion factor	0.001
F_{fish}	unitless	Fraction of fish contaminated	1
BW_{af}	kg	Body weight of adult female	66[c]
BW_i	Kg	Body weight of infant	9.4
$C_{milkfat}$	mg/kg-lipid	Concentration in milkfat	Calculated
h	days	Half-life of chemical	2555 (7 years) for PCBs
f_1	unitless	Fraction of ingested chemical stored in fat	0.9 for PCBs
f_2	unitless	Fraction of mother's weight that is fat	0.3
f_3	unitless	Fraction of breast milk that is fat	0.04
f_4	unitless	Fraction of ingested chemicals that is absorbed	0.9 for PCBs
ED_c	year	Exposure duration of breast-feeding child	1
EF_c	days/year	Exposure frequency of breast-feeding child	365 days/year
AT_c	days	Averaging time – carcinogen	25550 (70 years)[d]
AT_{nc}	days	Averaging time – non-carcinogen	= ED x EF
$ELCR_{child}$	risk	Excess lifetime cancer risk	Calculated
HQ_{child}	hazard	Hazard quotient	Calculated
SF_o	$(mg/kg/day)^{-1}$	Cancer slope factor – oral	2 for PCBs
RfD	(mg/kg/day)	Reference dose (chronic)	2×10^{-5} for PCBs
MRL	(mg/kg/day)	Minimal risk level (intermediate duration)	3×10^{-5} for PCBs

Notes:

a) Exposure assumptions taken from *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities* (EPA 530-R-05-006, September 2005), except as noted.
b) One of the higher ingestion rates used in the Portland Harbor risk assessment.
c) EPA combustion facilities guidance uses 70 kg (average weight of male and female adults).
d) EPA combustion facilities guidance is to use 1 year. We considered this too conservative, and used the lifetime AT_c value typically used at Superfund sites.

Attachment A

Derivation of Equation for Chemical Concentrations in Milkfat

The EPA combustion facility guidance document[1] and ATSDR's Toxicological Profile[2] do not elaborate on the derivation of the equation for calculation of chemicals present in milkfat. The main EPA reference for the equation is from an evaluation of infant exposure to chlorinated dibenzodioxins and chlorinated dibenzofurans in breast milk[3]. In this attachment, we explicitly derive the steady-state equation used to approximate chemical concentrations in maternal body fat, which is assumed to be equivalent to the concentration in breast milk.

The chemical body burden in the mother is calculated assuming first-order kinetics:

$$B_t = B_0 \, e^{-kt}$$

Where:

t = Time period (years)
B_t = Body burden at time t (mg)
B_0 = Body burden at time t = 0 (mg)
k = Rate constant = ln(2) / h (days^{-1})
h = Half life of chemical in body (days)

Using a similar approach, the maternal daily chemical intake, m (mg/kg/day), is used to calculate the concentration of chemical in the mother's tissue. The contribution to maternal chemical levels (C_{mother} in mg/kg-body-weight) is:

$$C_{mother} = \int_0^T m e^{-kt} \, dt$$

where the mother is exposed to chemicals in fish from time t = 0 to time $t = T$ (in days). The general solution to this equation is:

$$\int_0^T m e^{-kt} \, dt = \frac{m e^{-kT}}{-k} - \frac{m e^{0}}{-k} = \frac{m e^{-[\ln(2)/h]T}}{-k} - \frac{m}{-k} = \frac{m e^{-[\ln(2)][T/h]}}{-k} + \frac{m}{k}$$

$$= \frac{m\,(0.5)^{T/h}}{-k} + \frac{m}{k} = \frac{m}{k}\left[1 - (0.5)^{T/h}\right]$$

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[1] U. S. EPA. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* EPA 530-R-05-006, September 2005.
[2] ATSDR. *Toxicological Profile for Polychlorinated Biphenyls.* November 2000.
[3] Allan H. Smith. Infant Exposure Assessment for Breast Milk Dioxins and Furans Derived from Waste Incineration Emissions. *Risk Analysis*, Vol. 7, No. 3. 1987.

Substituting again for $k = \ln(2) / h$,

$$C_{mother} = \frac{mh}{\ln(2)} [1 - (0.5)^{T/h}]$$

If the exposure period of the mother to contaminated fish (T) is equal to the chemical half-life (h) of 7 years for PCBs, then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.5 \frac{mh}{\ln(2)}$$

If the exposure period of the mother to contaminated fish is equal to four half-lives ($T = 4h = 28$ years), then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.94 \frac{mh}{\ln(2)}$$

The limit of $[1 - (0.5)^{T/h}]$ for large values of T (relative to the half-life h) is 1. Therefore, at exposure periods to the mother longer than the chemical half-life, a reasonably conservative assumption is that the chemical concentration in the mother can be approximated by:

$$C_{mother} = \frac{mh}{\ln(2)}$$

This equation is further refined by considering the fraction of the chemical stored in fat tissue (f_1) and the fraction of the mother's weight that is fat (f_2).

$$C_{mother} = \frac{mh}{\ln(2)} \frac{f_1}{f_2}$$

Substituting the symbol ADD_{mother} for m, and assuming that the chemical concentration in milkfat is equivalent to the chemical concentration in the mother's lipid tissue, yields the equation for $C_{milkfat}$ shown in the main text.

$$C_{milkfat} = \frac{ADD_{mother}\, h}{\ln(2)} \frac{f_1}{f_2}$$